Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

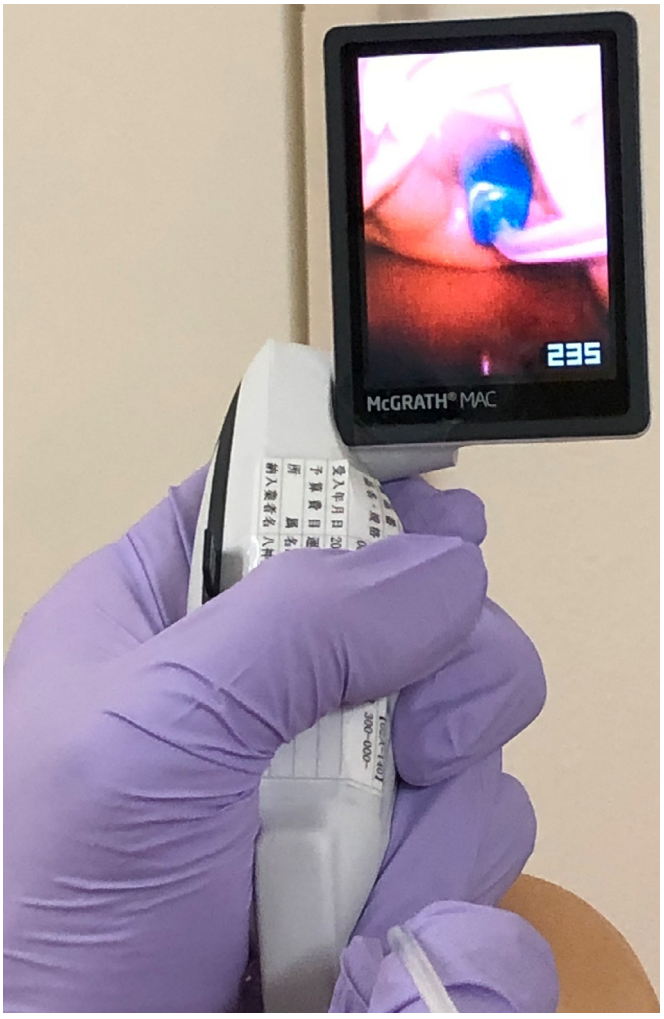

Fig 1. McGRATH MAC video laryngoscopy image showing a bronchial blocker passing through the glottis of a manikin.

disadvantage is the need for laryngoscopy. We believe that this method can be an alternative to DLT in the management of thoracic anesthesia. Currently, we are conducting a trial comparing this method and DLT for patients undergoing VATS and robotic-assisted thoracoscopic surgery.

Conflict of Interest

None.

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The 5 Reasons Why People Die of Coronavirus Disease 2019

To the Editor:

THE NUMBER of patients dying of coronavirus disease 2019 (COVID-19) exceeds by far the number of patients requiring admission to the intensive care unit (ICU). This is confusing for members of both the scientific community and the public. Here, we want to suggest and outline the reasons and locations of COVID-19 deaths, with the aim to clarify the issue.

Organ reserve might be impaired in some people as a result of elderly biologic age or comorbidities. For example, a 90-year-old patient with congestive heart failure or severe chronic pulmonary disease dies because even a mild lung involvement causes inadequate oxygenation and multiple organ failure. The median age of patients who died of COVID-19 was 82 years in Italy,[1] meaning that half of them were ≥82 years old. The only strategy to protect these patients is to prevent severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) exposure by means of self-isolation. These patients represent the majority of COVID-19 deaths; they usually remain in nursing homes or in hospital wards as a ceiling of treatment and are not admitted to the ICU.

The viral infection itself might be deadly because SARS-CoV-2 and its pathogenic mechanisms are known to be extremely dangerous.[2] Treatment with antiviral agents is limited because of the narrow time frame in which their administration could be helpful, making them difficult to use in routine clinical practice. Moreover, it still is not clear which drug could be superior to others in terms of effectiveness. Remdesivir is the only antiviral agent extensively tested and used for the treatment of COVID-19 in adults, but it is not life-saving in advanced clinical cases. In addition, its high cost and the need for intravenous administration interfere with its early use in the out-of-hospital setting. Several other antiviral agents have shown promising signs but have not been studied extensively so far (eg, umifenovir, daclatasvir-sofosbuvir, favipiravir). Patients in this group are relatively few and from all age groups.

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The disproportionate inflammatory response to SARS-CoV-2 is likely the cause of death of several COVID-19 patients as a result of acute respiratory distress syndrome and initial immunothrombosis. Corticosteroids and other immune suppressant agents might be used, with particular attention to the timing of administration. At the moment, steroids are used extensively to prevent this excessive inflammatory response, but they have proven to be ineffective or even dangerous when administered during the early phases of the disease[3] or to young patients.[4]

Microvascular COVID-19 lung vessels obstructive thromboinflammatory syndrome[5] can worsen hypoxia and cause death in a large proportion of patients. This syndrome consists of in situ pulmonary clot formation but does not exclude the classic thromboembolism. For this reason, thromboprophylaxis is an essential element for a favorable prognosis, and full anticoagulation is a mainstay of advanced treatment.

Complications of preexistent comorbidities or ongoing therapies (multidrug-resistant bacterial pneumonia or severe immune suppression) are other important, indirect causes of COVID-19 patients' deaths, together with rare acute clinical manifestations (eg, myocarditis).

In Italy, we estimate that patients dying in the ICU accounted for only 18% of the total number of deaths. The mean length of ICU stay was 15 days, with a 50% mortality rate[6] (529,946 bed-days were registered from February 21, 2020–February 21, 2021).[7] The number of ICU deaths was, therefore, 17,664 of 95,992 total,[8] equal to 18%. All other deaths have occurred at home, in healthcare facilities, or in hospital wards, according to each patient's characteristics.

These observations can help readers to interpret the numbers and figures daily distributed by the media.

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A Call for Pragmatic Bedside Assessment of Right Ventricular (RV) Function in Coronavirus Disease 2019 (COVID-19)

To the Editor:

We read with interest Isgro et al.'s[1] recent article, describing the potential for right ventricular dysfunction (RVD) in critically ill patients with coronavirus disease 2019 (COVID-19). We agree that the combination of micro/macro thrombi, myocardial injury, sepsis with a profound systemic inflammatory response, along with the combination of Acute Respiratory Distress Syndrome and injurious invasive ventilation, are likely to reflect a *perfect storm* of pathophysiology in which right ventricular (RV) dysfunction is highly likely to occur.

The authors suggest that RVD is present when echocardiography parameters (including RV fractional area change, tricuspid annular plane systolic excursion and pulsed-Doppler S'Wave velocity) are "less than the lower value of the normal range." While we wholeheartedly support the use of echocardiography as the cornerstone technique for assessment of RV function in this patient group, we call for a pragmatic approach that includes the combination of both qualitative and quantitative parameters.[2]

The quantitative parameters described by the author have not yet been validated in this population, either to a clinical endpoint or against a reference method. In other settings, they have been shown to be inconsistent for prediction of poor RV function when compared with reference methods, and perhaps more challengingly, have been observed to vary in their predictive performance depending on the degree of RVD present.[3]

Indeed, in the prospective study of 100 consecutive patients with COVID-19 presenting to the hospital by Szekely et al., even in the most critically ill cohort (those receiving invasive mechanical ventilation, n = ten), mean ± standard deviation values for pulsed Doppler S'Wave velocity and tricuspid annular plane systolic excursion were within the normal range (10.1 ± 3cm/s and 2.1±cm, respectively)[4]. Yet, in this cohort, RV dilatation (as measured by RVEDA) right ventricular end diastolic area was a common abnormality. Where quantitative parameters are used, their combination may allow better discrimination of normal and abnormal RV function.[5]

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